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THE FUSARIUM DISEASE (WILT) OF COTTON AND ITS CONTROL[1]

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INTRODUCTION

Cotton is the most important crop in Egypt, on which the prosperity of the country depends. In 1925 the total area in cotton was 1,924,382 feddans,[2] of which 1,128,946 feddans were in Sakel and 795,436 in Ashmouni and other varieties.

The Sakel crop for that year was estimated at 3,779,990 kantars, and the Ashmouni and other varieties at 4,080,939 kantars,[3] making a total of 7,860,929 kantars.

The average price of Sakel per kantar in 1925 was $35.78, and for the Ashmouni $25.06, making a total approximate value of $135,258,000 for the Sakel and $20,453,000 for the Ashmouni and other varieties.

So far the varieties most affected by Fusarium disease are those producing the long-staple cotton, of which Sakel is the principal and the one most grown in the Delta. Ashmouni and Zagora, two short-staple varieties grown mostly in Upper Egypt, are immune to this disease.

HISTORY

Atkinson (2) in 1882 was the first to investigate Fusarium disease in the United States. The fungus was isolated and described as a new species under the name *Fusarium vasinfectum.*

Erwin F. Smith (11) in 1899 described what he thought to be the ascogenous stage of the *Fusarium* of cotton wilt and called it *Neocosmospora vasinfecta* (Atk.) Syn. *Fusarium vasinfectum* Atk. He described it as follows: "Probably also on okra. Parasitism not proved. Genetic connection of various spore forms not proved. Chlamydospores not observed."

W. A. Orton (10) in 1900 produced the wilt disease "in healthy cotton plants by inoculating the soil in which they grew with pure cultures of conidial stages of *Neocosmospora vasinfecta.*"

[1] A detailed bulletin by the author is in the press. The author is indebted to Dr. E. C. Stakman for his assistance in preparing the manuscript.

[2] The feddan is 4,200 square meters.

[3] The kantar (unginned) is 141 kilograms.

Victor Mosseri (9) in 1902 was the first to diagnose this disease in Egypt and attributed it to *Neocosmospora vasinfecta* (Atk.) Smith.

Higgins (8) in 1909 in North Carolina and Butler (4) in 1910 in India have thrown some doubt on the parasitism of *Neocosmospora* and on its genetic connection with the wilt-producing Fusaria.

Other workers in the United States and elsewhere published short studies on this disease. The most important are those of Gilbert (7) in the United States, Ajrekar and Bal (1) and Butler (5) in India, and Briton-Jones (3) in Egypt.

DISTRIBUTION

A survey made during the cotton-growing seasons of 1924 and 1925 has shown that the disease is prevalent in the fertile parts of the Delta and absent in Upper Egypt.

In Lower Egypt the disease was found to be severe in certain localities, less so in some, and absent, as far as known, in other localities.

In Upper Egypt, where Ashmouni and Zagora, two varieties resistant to this disease, are grown, a peculiar localized discoloration has been observed within the central cylinder of certain plants of these varieties. This discoloration is much deeper in color than the characteristic one found in diseased susceptible varieties, such as Sakel cotton, and is localized in a way not found in typically diseased plants. From this discolored zone a species of *Fusarium* was isolated, but so far has not proved to be parasitic on Sakel or Ashmouni in spite of numerous inoculations. Besides, Sakel cotton—

TABLE 1.—*The occurrence of the Fusarium disease of cotton in Lower Egypt*

Province	District	Severity of attack	Province	District	Severity of attack
Gharbia	Tanta	Very severe	Menoufia	Ashmoun	Very severe
	Kafr El Zayat	do		Shibin El Kom	do
	Dessouk	Severe		Tala	do
	Zifta	do		Quesna	Severe
	Santa	do		Menouf	do
	Mahala El Kobra	do	Behera	Damanhur	Very severe
	Talkha	do		Abu Homos	do
	Kafr El Sheikh	do		Delingat	do
	Sherbin	Slight		Kom Hamada	do
	Foa	do		Teh El Baroud	Severe
	Belkas	Very slight		Shubrakhiet	do
Dakahlia	Simbilawin	Very severe	Sharqia	Belbis	Slight
	Aga	do		Hehia	do
	Mit Ghamr	do		Fakous	do
	Mansura	Slight	Qaliubia	Toukh	Slight
	Faraskour	Very slight		Qualuib	Very slight

which is very susceptible to this disease—was grown in fields where Ashmouni and Zagora cotton plants were previously found locally discolored, but in no case did Sakel become infected. It is therefore logical to conclude that at present this disease is absent in Upper Egypt and that this peculiar localized discoloration found in Ashmouni and Zagora is possibly due to one or more non-parasitic soil Fusaria which may enter the cotton plant through some mechanical or other injury, but which are unable to develop within the living plant tissue beyond a localized portion.

Table 1 gives the localities in Egypt where the disease was observed, as well as the severity of the attack.

FIG. 1. Early mosaic symptoms of Fusarium disease on cotton leaf.

ECONOMIC IMPORTANCE

The parasite attacks principally long-staple varieties of cotton. The disease is so severe in some parts of the Delta that many cultivators have given up growing Sakel cotton and have substituted short-staple varieties resistant to *Fusarium*. The conditions in Lower Egypt are most suitable for growing Sakel, the most important of the Egyptian long-staple cottons. It would be, indeed, a great loss to the country, if, as a result of Fusarium disease, the growing of this long-staple variety had to be abandoned.

At present, Egypt produces the bulk of the long-staple cotton of the world. Much of the prosperity of the country depends on this fact. Since the great war, many other countries have started cotton growing on a large scale, but they will probably produce cotton of a shorter staple than the Egyptian long-staple varieties. It seems to the author, therefore, that to maintain the present prosperity of this country the production of long-staple

cotton must be kept up. To realize this, the Fusarium disease must be controlled in a way that will not interfere with the production.

Happily this work has led to the isolation of four strains of Sakel which are perfectly resistant to this disease and of good long-staple quality. This matter will be considered later in this article.

DESCRIPTION OF THE DISEASE

The name "wilt" for the Fusarium disease may suggest that the wilting of the diseased plant is a necessary symptom. Under Egyptian conditions such is not the case. Infected plants may appear externally healthy, and

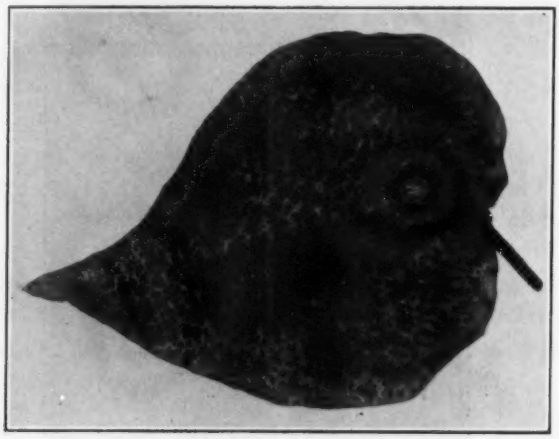

FIG. 2. Later symptoms of Fusarium disease of cotton, in which the entire leaf surface is mosaic.

yet on examination of the roots the characteristic discoloration can be easily observed in the tissue of the central cylinder. For this reason "Fusarium disease" is suggested as a name for the disease as it occurs in Egypt.

External Symptoms: the mosaic symptoms of the leaf. The very characteristic symptom is the appearance of a yellowish network on the leaves. This mosaic, as it is called, starts generally in one corner of the leaf (Fig. 1) and extends until it may cover the whole leaf surface (Fig. 2). It is apparent on both sides of the leaf, but mostly on the upper surface. The affected part of the leaf may or may not dry up in the former case; it turns light brown.

This mosaic symptom appears more frequently in the seedling stage than in the adult plants. It is of very frequent occurrence in susceptible cotton seedlings grown in infected soil in pots. Its frequent occurrence seems to be associated with abundant soil moisture.

Although the mosaic has been observed as a sure sign of the disease, its association with diseased plants is not constant, and for this reason its absence does not indicate freedom from disease.

External Symptoms: the death of the stem. The stem of a diseased plant may die, starting at the growing point and proceeding downwards. The whole plant may die or may survive by giving rise to new shoots from the lower portion of the stem (Fig. 3, A). The newly formed shoots may

FIG. 3. A. Cotton plant in which the growing point has been killed by *Fusarium*. New shoots have grown out from the lower portion of the stem. B. Infected cotton plant which is very much dwarfed and has produced numerous basal branches.

in turn show signs of the disease. Later, however, the recovered plants may be dwarfed, with much basal branching and poor productive capacity (Fig. 3, B).

Internal Symptoms. The roots of a diseased plant which has passed the seedling stage always show a characteristic discoloration on being cut longitudinally. The discolored tissue is dark olive green and is arranged in irregular "bundles of streaks" (Fig. 4). This discoloration, which is in the central cylinder of the plant, may extend from the root system upwards. It may reach the growing point and even the boll-stalk. Its presence in the boll-stalk as a possible source of infection of the seed is discussed later.

THE CAUSAL ORGANISM

The Parasite within the Host. When hand sections of the roots of an infected plant are examined microscopically, hyphae of the parasite are found within some of the xylem vessels in the discolored tissue. These hyphae may be so crowded together as to plug some of the vessels or may be so sparse that only a few can be observed (Fig. 5).

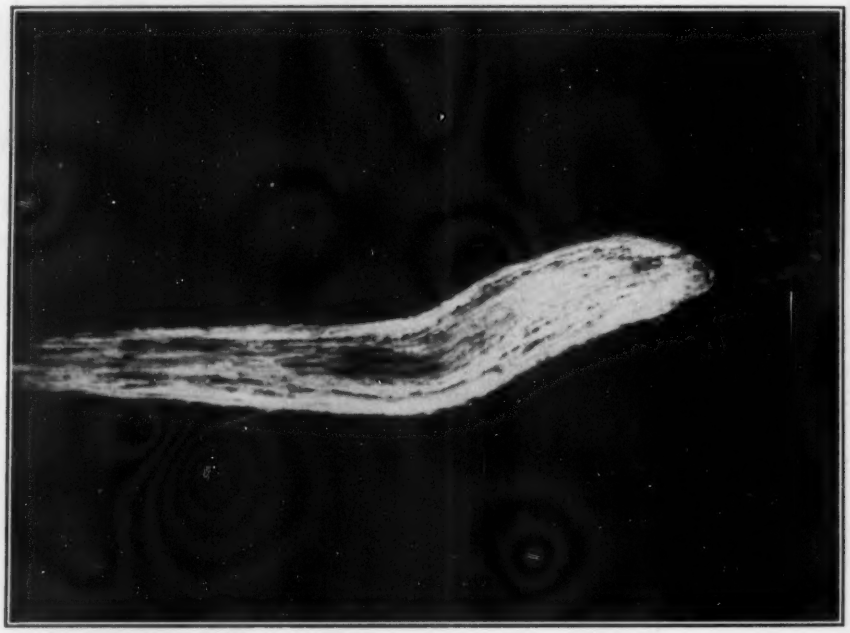

FIG. 4. Root of a cotton plant split to show the discolored areas characteristic of Fusarium disease.

The hyphae may be creamy white or of a yellowish tinge as if externally coated with a pale yellow gum-colored substance. The walls of the xylem vessels may also be stained a deeper yellow. The same yellowish coloring matter may also fill some of the smaller cells near the invaded xylem cells. Other cells may also be filled with a very dark gum-like substance. Tyloses

have also been observed. So far no spores have been found within the invaded cells.

During the life of the plant the parasite seems to limit its invasion to the xylem vessels; at the death of the host plant, however, and if it is kept under moist conditions, the parasite appears on the surface of the infected portion as tufts of mycelium bearing myriads of spores.

Isolations. The fungus is easily isolated from diseased plants by placing short lengths of peeled infected roots or stems under aseptic conditions on any suitable medium. The fungus, which is a species of *Fusarium*, appears generally within a week as white mycelial tufts, either from one or both of the cut ends, or from the peeled surface. At first the tufts consist of hyphae with few microconidia, later macroconidia are produced, and then chlamydospores.

Parasitism. Isolations were made from diseased plants obtained from infected fields in the different provinces of Lower Egypt, where the disease is known to be severe. The fungus was transferred on acid potato plug, on which it grows vigorously; after 10 days' incubation at 30° C. these cultures were mixed with a weighed amount of soil known to be free from *Fusarium*, and Sakel and Ashmouni cotton were sown in it. The fungus in all cases proved to be parasitic on Sakel, while Ashmouni cotton was immune. The parasite was re-isolated from the diseased plants and proved again to be parasitic on Sakel.

The Morphological and Cultural Characters of the Fungus. A single spore culture was obtained and tested for its pathogenicity. The culture developed normally, and produced all forms of spores, microconidia, macroconidia and chlamydospores. It was examined after 40 days at 30° C.

Microconidia: 0–3 septate, typically 0 and 1 septate, majority ellipsoidal, sometimes slightly curved, few globose to pear-shaped, rarely comma-shaped.

Macroconidia: 0–5 septate, typically 3 and 4 septate, sickle-shaped to straight, gradually attenuate more towards the apex, pedicellate.

Chlamydospores: Subglobose to spherical, generally smooth and thick-coated; terminal, 0–1 septate, formed on or in mycelium; intercalary, single or in chains, rarely in clusters. In old cultures and under reduced aeration, conidial chlamydospores develop, mostly in macroconidia.

Mycelium: Hyphae copious, slightly branched, some very granular, varying greatly in diameter (2.5 to 5 μ. Septation varying in size. The hyphae appear colorless under the microscope, but in mass, when grown on acid potato plug, the mycelium is not dead white but has a very faint bluish hue and is cottony in appearance. Anastomosis occurs, but is rather rare.

No sclerotia have been observed up to now.

Spore germination: At room temperature (25° C.) microconidia and macroconidia germinate readily in water. Some of the chlamydospores germinate while others do not. The drop cultures were kept under observation for one week.

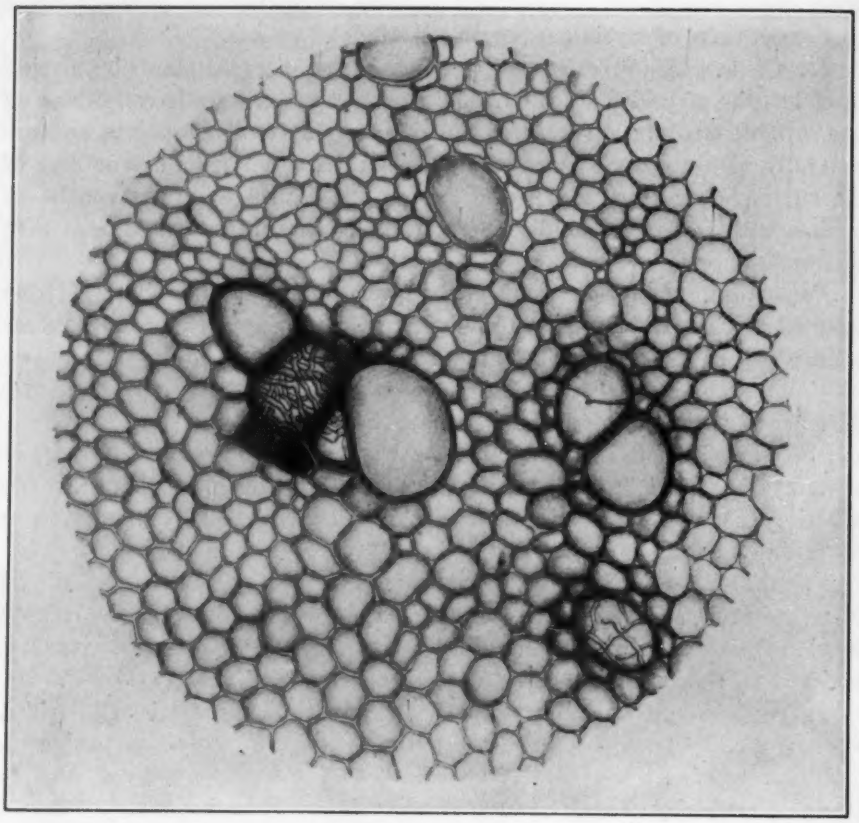

FIG. 5. Cross section of a cotton root showing the hyphae of *Fusarium* in the xylem vessels.

The Growth of the Fungus on Different Media. On standard liquid media[4] the fungus grows best on those containing 3 per cent glucose: when the same percentage of saccharose is substituted, growth is slightly less.

If 1.5 per cent citric acid is used instead of either of the sugars, there is less mycelial growth but a great development of chlamydospores. On

[4] The standard medium, as used by Butler (4), was made up as follows:

Ammonium nitrate	10	gms.
Potassium phosphate	5	gms.
Magnesium sulphate	2.5	gms.
Distilled water	1000	cc.

standard medium, to which sodium carbonate has been added, growth is very poor.

On solid media the fungus grows best on acid potato plug (lactic acid).

The Relation between Host and Parasite. The author, in a previous paper (6), studied the production by *Fusarium solani* (Mart) Sacc. of a toxic substance capable of causing wilting in plants. The excretory substance of the Egyptian *Fusarium* of cotton was similarly studied. It was found that when the fungus was grown on modified Richards' solution a staling substance was formed which was capable of causing cotton seedlings to wilt when these were placed in the filtrate of the culture. This staling substance increased with the age of the culture. The alkalinity of the filtrate of the culture also increased with the age.

It is possible that the parasite within its host is capable of producing similar staling substances which may under certain conditions cause the infected plant to wilt.

The Temperature Relation. The optimum temperature for the growth of the fungus on acid potato plugs was found to be between 30° and 35° C.

The incubation period, the time required for the mosaic symptom to appear after sowing susceptible cotton seed in infested soil, varies with the average day temperature. Sakel cotton was grown in uniformly infested pots in February, April, June, July, August and October. Table 2 gives the results.

TABLE 2.—*The effect of the average day temperature on length of incubation period with cotton plants grown in Fusarium infected soil*

Date of sowing, 1924	Average day temperature during growth period in degrees C.	No. days required for mosaic to appear after sowing
Feb. 2	15.9	58
Apr. 18	23.7	14
June 18	26.1	14
July 23	26.9	12
Aug. 28	25.0	14
Oct. 20	20.6	22

It is worth noting that under field conditions, when cotton is sown in March, the first symptoms appear from 60 to 90 days after sowing.

SUSCEPTIBILITY OF COTTON VARIETIES

Egyptian Varieties. The different varieties of Egyptian cotton vary in their susceptibility to this disease: Sakel cotton is the most susceptible,

while Ashmouni and Zagora (a selected strain from Ashmouni) are immune. Table 3 gives the average infection obtained in a number of trials with several Egyptian varieties. The estimation of susceptibility was calculated on

TABLE 3.—*The susceptibility of Egyptian varieties of cotton to Fusarium disease*

Variety	No. of plants			Percentage	Variety	No. of plants			Percentage
	Total	Healthy	Diseased			Total	Healthy	Diseased	
Sakellaridis	88	3	85	96	Casuli	131	103	28	21
Garofalou	80	6	74	92	Afifi	44	35	9	20
310	189	20	137	73	Toudri	282	236	46	16
Assili	244	111	133	54	Pilion	307	266	41	13
Nahda	60	30	30	50	Fathi	194	181	13	6
Nubari	136	104	42	30	Ashmouni	489	489	0	0
Abbassi	87	54	23	26	Zagora	259	259	0	0

the basis of the number of plants which were found typically discolored in the central cylinder of their roots when two months old, plus the number of plants which had died at an early age from the disease. The parasite was isolated from all of these.

Susceptible Age of Host. Infection is possible at any time during the life of a susceptible cotton plant as long as its root comes in contact with the parasite. The seedling stage is, however, the period when injury is most likely to be fatal.

During the warmer part of the year, when seedlings are grown in heavily infected soil, the parasite produces a distinct brick-colored zone at the hypocotyl, somewhat similar to that caused by sore-shin. The seedlings are girdled at that point, thus causing the plants to collapse and gradually dry up.

Other Hosts. The Egyptian Fusarium parasite of cotton is specialized in its parasitism not only for cotton, but for certain strains of cotton.

The Mode of Infection. The actual mode of infection is under study. Investigation has shown, however, that the parasite is able to enter the root system of its host unaided by other soil organisms. When injected hypodermically into the stem of a healthy, but susceptible cotton plant, it is able to develop and produce the typical discoloration in the central cylinder at the part injected. When, however, immune cotton plants are similarly injected, the parasite is unable to develop, but produces a localized discolored portion not typical of the disease. In both cases, however, the parasite was reisolated and proved to be parasitic.

Is the disease seed-borne? Under Egyptian conditions up to now, the pathogene does not seem to be seed-borne. Trials over two years were carried out. Seed from heavily infected plants were grown in sterile soil. The seed were divided into two lots; one lot was externally sterilized by delinting in concentrated sulphuric acid, the other sown without disinfecting. In both cases none of the 145 plants grown and allowed to mature developed any external or internal symptoms of the disease. The author also failed to isolate the parasite from the seed of heavily infected plants, which were treated as above. This investigation is being continued.

THE RELATION BETWEEN THE DISEASE AND THE SOIL

The infective capacity of the soil was found to vary within the same field. The disease first appears in the field in patches. With the frequency of growing susceptible cotton the patches increase in area until finally the whole field may become involved. Within these patches the cotton plants die at an early age. In the immediate area surrounding the patches, plants of the same susceptible variety show no definite, apparent signs of the disease, yet when they are pulled up and their roots slit, the characteristic discoloration is found in the majority.

In less infective fields with susceptible varieties of cotton the plants may appear at first sight as healthy. However, when their roots are examined, many are found to be typically infected.

Investigations carried out at Giza have shown that the different degrees of infective capacity of the soil vary with the amount of infecting matter present either through its accumulation year after year, due to the growing of susceptible cotton, or by the addition of different amounts of the organism in culture.

Type of Soil. The soil found infected most frequently is that of heavy texture, contrary to what occurs in the United States where wilt is most prevalent in light soil.

In the Delta the disease is most severe in the heavy fertile land. Experimentally, the attack was more severe with the addition of organic manure to infected land.

Investigation has shown that the fungus may be found as deeply buried in infected fields as one meter and perhaps more. The parasite is probably carried into the deeper layers with the growth of infected roots of the host. Infection is most severe, however, in the first 40 centimeters below the surface.

Observations over a number of years have suggested that in the case of the well distributed soil infection, similar to that which occurs under natural field conditions, the plant is subjected to many repeated infections, as its new roots grow into the soil containing the parasite.

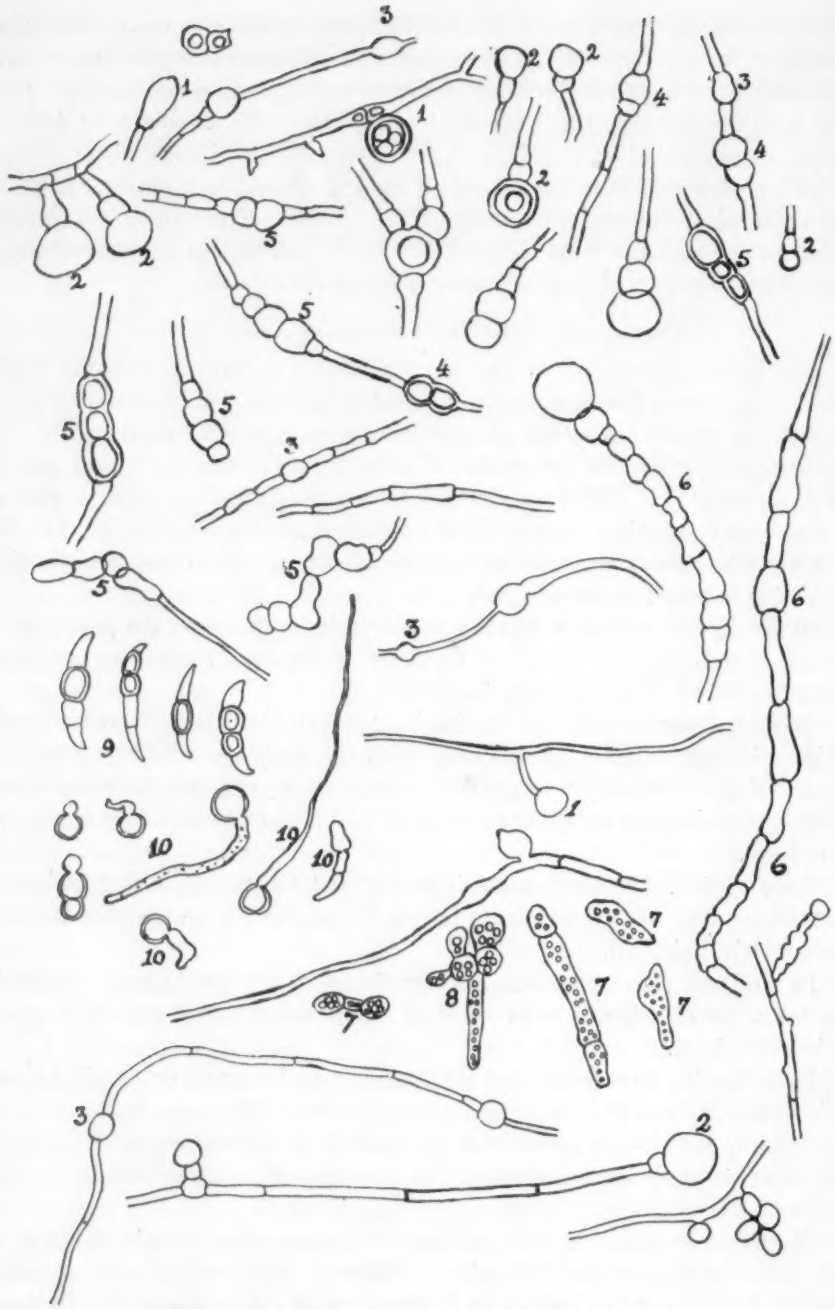

FIG. 6. Various stages of the Egyptian *Fusarium*.

However, in case the soil is artificially infected only 20 or 30 centimeters below the surface, many plants are subjected to attack only in their early stages and later the roots grow into the deeper clean soil layers. Many plants arrest the spread of the parasite within their tissues by forming a gummy substance about the local infections.

CROP ROTATION AND SEVERITY OF DISEASE

A rotation experiment started at Gemaiza farm in 1922 has shown that up to 1926 the severity of disease increases with the frequency of susceptible cotton crops.

On six plots planted with Sakel cotton in 1923, 1924, 1925 and 1926, the average percentage of infected plants in 1926 was 74.5.

On six plots planted with Sakel cotton in alternate years, 1923 and 1925, the average percentage of infected plants in 1925 was 25. On six similar plots planted with the same variety in 1924 and 1926 the average percentage of infected plants was 33.

When Sakel cotton was grown every third year, the average percentage of infected plants was 10 on one series of six plots and 23 in another series of six plots.

THE RELATION BETWEEN THE EGYPTIAN FUSARIUM, THE INDIAN FUSARIUM, AND FUSARIUM VASINFECTUM

To attempt to identify the Egyptian *Fusarium*, it was necessary to compare it with the Indian and American fungi producing wilt diseases in the respective countries.

The morphological differences between the microconidia and macroconidia of these three fungi is too small to be of any comparative value. There are, however, some differences in the morphology of the chlamydospores, substratum coloration, and cultural characters of these respective fungi.

Chlamydospores. The three fungi were grown under identical conditions on acid potato plug and examined after 40 days' incubation at 30° C.

In the case of the Egyptian *Fusarium* there is a tendency for the chlamydospores to become smaller towards one or both extremities of the chain in which they are borne. Dumb-bell-shaped chlamydospores often are produced (Fig. 6).

In the case of the Indian *Fusarium*, the basal cell of the 1-septate terminal chlamydospore is rectangular in shape and definitely constricted at the middle (Fig. 7).

The American *Fusarium* has a peculiar knotted appearance owing to the fact that some of its hyphae contain spherical to globose non-septate inter-

calary chlamydospores and that there is variation in the size of the chlamydospores in some of the chains (Fig. 8).

Substratum Coloration. The substratum coloration of the Egyptian *Fusarium* is very distinct from the Indian and American Fusaria.

Table 4 gives the different substratum colorations produced by the respective Fusaria.

TABLE 4.—*The substratum coloration produced on rice and on oatmeal agar by the Egyptian, Indian, and American strains of cotton Fusarium*

Culture of *Fusarium*	Medium	Substratum coloration
Egyptian	Rice	Brilliant yellow with light cobalt violet in parts.
Indian	do	Light cobalt violet, madder lake in patches, mineral violet ring.
American	do	A ring of deep mineral violet.
Egyptian	Oatmeal agar	Traces of permanent mauve of different shades.
Indian	do	Combination of cobalt violet and mauve.
American	do	Combination of deep mineral violet and light mauve.

Cultural characters. There are only slight differences in the three fungi as to their cultural characters. The American *Fusarium* grows better on some media than the other two. Under reduced aeration and in old cultures, the macroconidia and microconidia of the Egyptian and Indian *Fusaria* form conidial chlamydospores. In the American strain, however, up to now no such chlamydospores have been observed.

The parasitism of the three fungi. The Egyptian *Fusarium* is a virulent parasite on the long-staple Egyptian cotton and the Sea-Island varieties. It also is capable of attacking some of the Indian varieties.

The Indian *Fusarium* is non-parasitic on both the American and the Egyptian varieties, but parasitic on some of the Indian varieties.

The American *Fusarium* is a very weak parasite on the Egyptian varieties, non-parasitic on the Indian varieties tried, but parasitic on some of the American cotton varieties.

The above results were obtained after a series of trials carried out at Giza over a number of years. It is possible, however, that soil and climatic conditions at Giza may not be so favorable for the attack of the Indian and American parasites as the conditions prevalent in the respective countries where they produce the disease. Therefore the above results must be taken with certain reserve.

Moreover, in Egypt this disease is prevalent in heavy soil independently of the attack of *Heterodera radicicola*, while in the United States it occurs in light soil and is often associated with the root-knot disease.

The writer, in the light of the above description and behavior of the three fungi, considers that the Egyptian *Fusarium* of cotton is distinct from the American and Indian Fusaria producing the same disease in the United

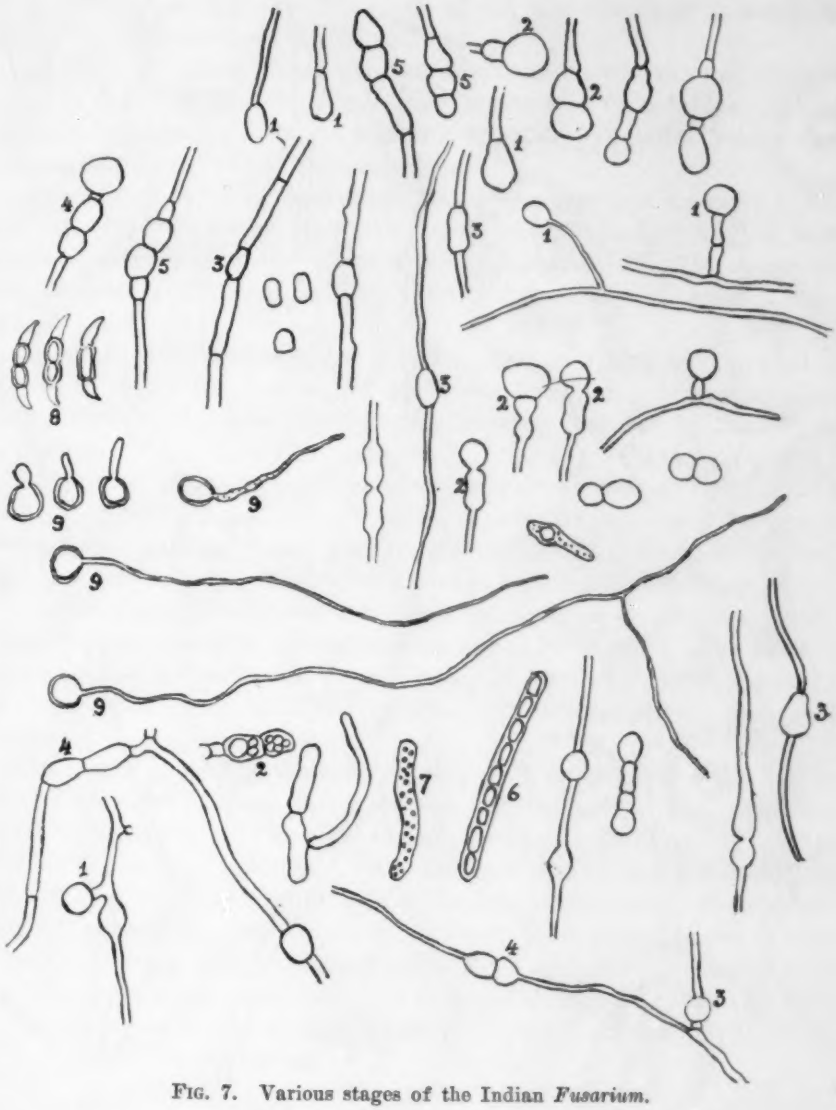

FIG. 7. Various stages of the Indian *Fusarium*.

States and India, and suggests calling this fungus, at any rate until further studies are carried out, *Fusarium vasinfectum* var. *egyptiacum*.

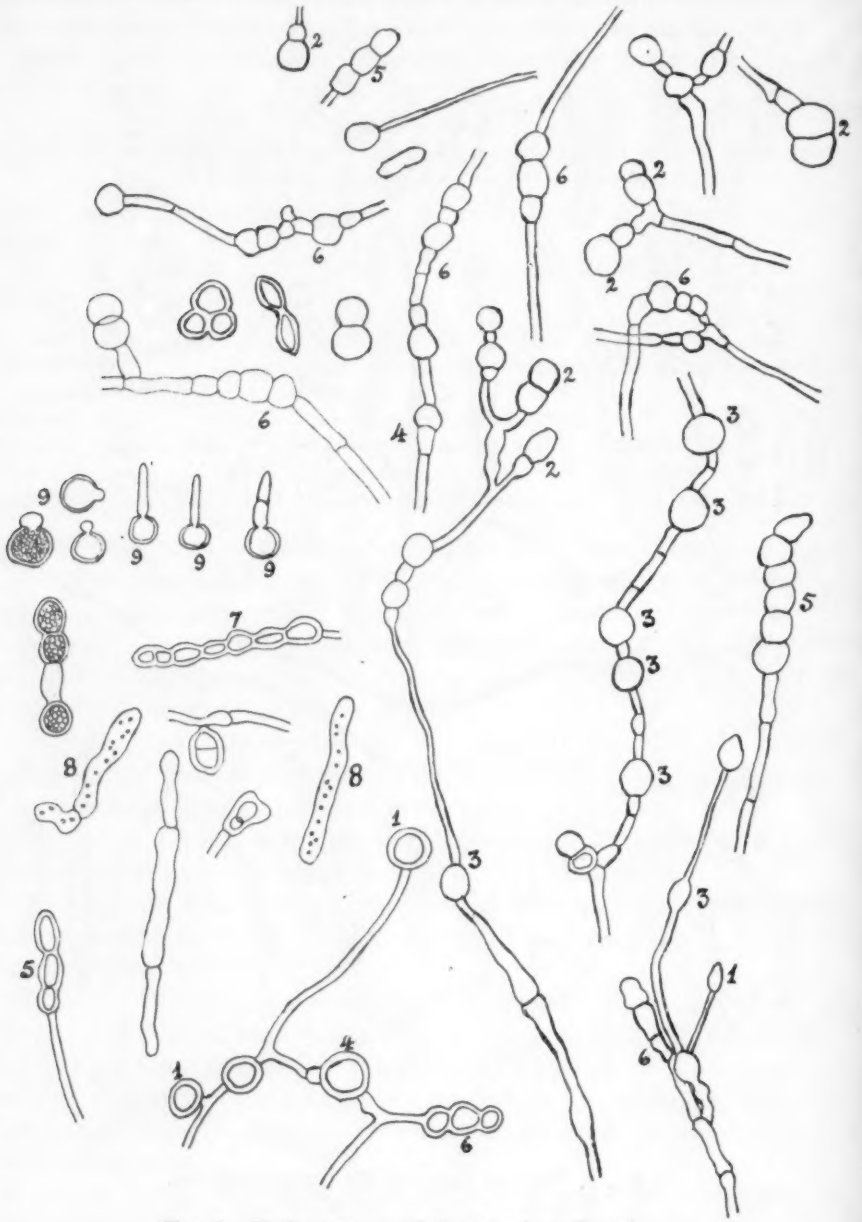

FIG. 8. Various stages of the American *Fusarium*.

CONTROL

The study of the control of the disease was divided into three parts: (a) the effect of the bare-fallow, (b) the disinfecting capacity of carbon bisulphide, (c) the selection of resistant long-staple cotton (Sakel).

Bare-fallow, and carbon bisulphide as soil injection, were found to be unsatisfactory as methods of control.

In 1923, 141 single plants were selected from heavily infected fields in Dakahlia and Menoufia provinces. These plants were from fields sown with Domains Sakel where infection was so severe that only a few plants were left standing by the end of the cotton season.

The progeny of the selected single plants were grown in heavily infected soil. Of these, plants of pure Sakel type which remained healthy were selected and sown again the following year in infected soil. The operations were repeated in 1925 and 1926 until finally four strains of resistant Sakel were obtained.

The lint of the mother-plants grown in 1925 and 1926 were graded as good Sakel by Mr. Charles Ross, of the British Egyptian Company, at Alexandria, and official grader of the Cotton Research Board of the Ministry of Agriculture.

The soil in which the selected cotton was grown was obtained from a heavily infected field and large quantities of parasitic cultures of the fungus were added. This soil was so heavily infected that 96 per cent of the seedlings of the ordinary Domains Sakel grown in it died from the disease.

To ensure complete exposure of the selected plants to infection, the infected soil was placed in bottomless pots buried in the field. The roots of the plants had in this way to grow through 50 centimeters of very heavily infected soil. Finally, in 1926, four strains of resistant Sakel cotton were obtained.

The seed of these four strains were enough to sow an acre and a half in 1927. Pure lines of these were grown in infected soil at Giza. The best plants of each strain will be selected and sown again the following year at Giza under the same conditions, while the bulk seed of each strain will be propagated at the experimental farm of Gemaiza or elsewhere. These operations will be repeated yearly, the nucleus plants being always grown at Giza in heavily infected soil, while fresh lots of bulk seed of each strain will be sown for propagation every year. In this way there will, eventually, be a continuous yearly flow of resistant Sakel seed from Giza through the propagation farms to the cultivators.

SUMMARY

1. The Fusarium wilt of cotton is so destructive to long-staple cotton in some localities of Lower Egypt that growers have been forced to sub-

stitute less valuable short-staple varieties which are resistant.

2. The prosperity of the country depends largely on the successful production of long-staple cotton; therefore it is essential to attempt to control the wilt.
3. The symptoms of the disease in Egypt and the morphology and some of the physiological characteristics of the causal organism are described.
4. In modified Richard's solution the pathogene produces a staling substance which causes cotton plants to wilt.
5. The optimum temperature for the growth of the fungus on acid potato plugs was 30°–35° C. The incubation period of the pathogene in the host varies with temperature, ranging from 58 days at 15.9° C. to 12 days at 26.9° C.
6. The pathogene seems able to penetrate cotton roots unaided by other soil organisms. It does not seem to be seed-borne in Egypt.
7. Heavy soil to which organic manure has been added is likely to be most heavily infected.
8. A comparative study was made of Fusaria causing cotton wilt in Egypt, India, and North America, respectively. There were some morphological and cultural differences. The three differ considerably in pathogenicity. The Egyptian form is virulent on long-staple Egyptian cotton, Sea-Island varieties, and can attack some of the Indian varieties; the Indian form is parasitic on some Indian varieties but not on American and Egyptian varieties; and the American form is non-parasitic on the Indian varieties tested, very weakly parasitic on Egyptian varieties, and parasitic on some American varieties.
9. Neither bare fallow nor the application of carbon bisulphide to the soil controlled the disease.
10. Four wilt-resistant strains of Sakel cotton were obtained by selection.

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A GLOEOSPORIUM BLIGHT OF RASPBERRY

B. O. Dodge

Orange-rust, cane blight, wilt or blue-stem, and spur blight are the best known fungous diseases of raspberries which are more or less systemic, and which result in the death of the affected laterals or main canes. For several years the writer has been observing some of the fungi which occur on different species, varieties, and new hybrids of *Rubus* in the breeding grounds of the Office of Horticulture at Bell, Maryland. One of the fungi most frequently found in early spring on the dead or dying canes of the previous season's growth is one which is probably the spur blight fungus. In certain morphological features, however—the presence of paraphyses in the ascocarp and the development of a pycnidial stage—the fungus does not altogether agree with previous descriptions of *Mycosphaerella rubina*. Spur blight is at first local in its attacks. The fungus may be found later throughout the length of the canes which it has killed. Sackett[1] has illustrated the early stages of this disease in his report. He has called attention to the gray patches that appear in the brown discolorations, and has noted that the black bodies seen on the gray patches are young ascocarps. He has not, perhaps, sufficiently described the appearance of the affected canes in the following spring, when ascocarps develop in great abundance over the entire surface, to the exclusion of most other fungi. In the spring the canes have a gray appearance. Such canes are likely to be very light and brittle and with but a limited amount of wood formed the previous season.

There is another disease which has also caused considerable damage to the red raspberry hybrids each year in the breeding plats at Bell, Md., and it is one which is being found in commercial plantings of black raspberries in Kentucky, Ohio, Michigan and other states. The writer is calling this disease Gloeosporium blight for reasons which will appear later. On the Cumberland variety it first causes a blackening or necrosis of the leaf stalks and tips of young shoots. Later the leaves collapse and the tip ends of the shoots turn purple or blue, the discoloration proceeding from the tip downward. The lower part of the young cane may remain green for some time. The whole turion may finally die or the disease may be confined to a single lateral. On account of the fact that the young canes, when badly infected, turn blue or purple the disease is likely to be confused during the summer season with the raspberry wilts which are known as blue-stem. In case of

[1] Sackett, W. G. Spur blight of the red raspberry caused by *Sphaerella rubina*. Colo. Agr. Exp. Sta. Bul. 206. 1915.

the raspberry wilts, the blue discoloration appears first at the base and works upward; while in the Gloeosporium blight the blueing of the shoot begins at the tips and works downward.

Spur blight first causes a brown discoloration of the affected parts. Later, after the perithecia have developed abundantly, the canes take on a gray appearance. It is in winter or early spring that the canes attacked during the previous summer by the Gloeosporium blight fungus present an appearance very similar to that of canes attacked by the spur blight organism. The entire cane may be infected (Plate XXVIII, A), or there may be only a broad, white or light gray streak of dead tissue along one side (Plate XXVIII, B); or there may merely be a dead area (Plate XXVIII, C) at the base of a dead lateral. Infected canes become girdled and eventually die without producing fruit, and in most cases without producing leaves.

A chromogenic *Gloeosporium* has been repeatedly isolated from the black oval spots on the gray canes each spring since 1922. Perithecia have been found in only one culture from all of this material. This culture, contaminated with bacteria, was obtained by transfer of a mass of conidia directly from an acervulus on a dead cane of the previous summer's growth. Pure cultures were made from this culture by the plating method. The new cultures happened to be all non-chromogenic. However, other sub-cultures from the original chromogenic culture on which there were ascocarps are invariably chromogenic. This seems to indicate that the original culture may have contained two different strains of *Gloeosporium*, one chromogenic and the other non-chromogenic. Ascocarps have developed in most of the new cultures of the non-chromogenic strain.[2] Shear and Wood[3] report the appearance of *Glomerella cingulata* on diseased canes of blackberry received from Shelbyville, Tennessee, in March. Typical acervuli of the *Gloeosporium* stage developed on these canes soon after they had been placed in the damp chamber. Later many perithecia of *Glomerella* matured on this material. In cultures which were obtained from the discolored wood of the diseased canes and grown on cornmeal agar in tubes, mature perithecia were produced. These cultures were all non-chromogenic. The writer has obtained about 50 single ascospore cultures from the non-chromogenic strain from raspberries, which to date have remained non-chromogenic. All the other cultures made from the fungus on red raspberry hybrids at Bell, Maryland, and Rosslyn, Virginia, have been chromogenic.

[2] The perithecial stage has since been found on raspberries from several different states.

[3] Shear, C. L. and A. K. Wood. Studies of fungous parasites belonging to the genus *Glomerella*. U. S. Dept. Agr. Bur. Plant Indus. Bul. 252. 1913.

Dr. Shear, who has examined not only the diseased raspberry material from Maryland, Virginia, Kentucky and Michigan, but also the various cultures obtained from these sources, informs the writer that he has been unable to find any morphological differences by which the raspberry strains can be distinguished from those commonly found on apples and referred to as *Glomerella cingulata* or *Gloeosporium cingulatum*. Shear and Wood have previously reported extensively on the great variability to be found in strains of this species as they occur on different hosts. They have made a large number of cross inoculations, which furnish evidence that the species frequently occurs on a wide range of hosts.

Infection of raspberry leaf stalks (Plate XXVIII, D) and shoots (Plate XXVIII, E) can readily be obtained either in the greenhouse or in the field by puncturing the cortex with a needle bearing conidia of the chromogenic strains. Within a very few days after making the inoculation, the cortex surrounding the puncture begins to shrink and turn brown or black. Within a week acervuli begin to develop (Plate XXVIII, E). New cultures from spores in acervuli on these artificially infected areas are chromogenic. When leaf stalks are punctured an inch or so from the base in making the inoculation, the mycelium spreads up and down fairly rapidly (Plate XXVIII, D), so that within a few days the leaf dies and falls. The fungus also may develop on leaves under certain conditions. When shoots were sprayed with spore suspensions, portions of several leaves turned brown and developed acervuli. These leaves may have been torn or scratched by thorns in the process of the experiments so that a way was opened for the invasion by the fungus. No evidence has yet been obtained that uninjured healthy raspberry shoots can be infected by merely spraying them with spore suspensions.

Only a limited amount of experimental work has been done with a view to determining whether bitter-rot strains of *Gloeosporium cingulatum* isolated from rotting apples will infect raspberries. Dr. Shear furnished the writer with a chromogenic and non-chromogenic strain from apple in order that this point might be tested out in a preliminary way. Leaf stalks and young growing canes of two different varieties of raspberry were inoculated with these strains by puncturing with a needle bearing conidia and then covering the wound with paraffin. Other canes were inoculated by forcing spore suspensions into young canes by means of a hypodermic needle. In some cases both of the strains from apples seemed to invade the living tissues of raspberry and form acervuli in about the same way as did the chromogenic strain from raspberry. One black raspberry shoot was punctured in nine different places. Infection followed in eight of the nine, and failure in the ninth may have been due to the fact that the wound had not been stopped with paraffin.

The raspberry and apple chromogenic strains developed at about the same rate and produced the same type of rot when inoculated into apples. The effects produced when apples and raspberries (Plate XXVIII, F) were inoculated with the non-chromogenic strain from apple were perhaps slightly different from those produced by the chromogenic strains referred to. There is no question in either case of the pathogenicity of the three strains on both hosts.

Material which Dr. W. D. Valleau has sent the writer from the Agricultural Experiment Station grounds at Lexington, Kentucky, indicates that certain varieties of black raspberry are very susceptible to infection by *Gloeosporium cingulatum*. On arrival the blackened tips of the young shoots (Pl. XXIX, A, b) showed masses of pink acervuli. Uncontaminated cultures were easily obtained by transfer of conidia directly from the acervuli on these blackened canes. When the canes were opened by splitting, one could see white mycelium penetrating the pith throughout the length of the blackened ends (Plate XXIX, B). The pith had turned brown or black and had become filled with pockets, due to drying out and cracking in sections. Many black raspberries were inoculated with the fungus from this material and all became infected.

In the Gloeosporium blight material from Kentucky and Michigan anthracnose (*Gloeosporium venetum*) could be seen in all stages of development, from the very earliest browning of small elevations surrounding the prickles to lesions with mature spores of anthracnose. Whether or not anthracnose very generally enters the young tissues of raspberry through hairs or through the primordia of prickles or thorns, there can be no question that most of the lesions in this material developed at the points of origin of prickles on the growing shoot tips. *Gloeosporium cingulatum* quickly established itself so that the anthracnose lesions contained acervuli of *G. cingulatum* as well as spores of *G. venetum*. Roberts[4] has pointed out that the apple bitter-rot strain of *G. cingulatum* may overwinter by living in cankers and other lesions in apple trees caused by other fungi. As attempts to infect the raspberry by spraying uninjured canes with spore suspensions have been without positive results so far, it may be assumed that the fungus does not penetrate the unbroken epidermis under ordinary growing conditions. In one field in Michigan, where the Gloeosporium blight was fairly abundant, much of it was found on plants affected with yellow mosaic. Dr. C. W. Bennett, in calling the writer's attention to this association, suggested that a plant infected with this virus disease may in some way be predisposed to attack by this blight fungus.

[4] Roberts, J. W. Sources of the early infection of apple bitter-rot. Jour. Agr. Res. 4: 59–64. 1915.

Just how much anthracnose, *Gloeosporium venetum*, may ordinarily be a factor contributing to the original infection of the raspberry canes by *G. cingulatum* can not be stated definitely here. All of the material sent by Valleau had anthracnose lesions on the green shoots and laterals (Plate XXIX, A, a). In most cases, as noted previously, these lesions contained acervuli of *G. cingulatum* in addition to the spores of the anthracnose organism. After photographing this material, the branch "a" (Plate XXIX, A) was cut off and the end placed in water. At that time the branch was turgid and green except where spotted with the anthracnose. Two days later the entire tip had become blighted and purplish black, and acervuli were breaking out through the cortex over the entire length of the infected portion. When this branch was split open it was found that the pith had also turned brown and had become pocketed to within two inches of the base (Plate XXIX, C). Evidently the blight fungus had spread very rapidly from each of the anthracnose lesions.

While Gloeosporium blight of raspberries in Maryland and Virginia has not been so serious as to interfere greatly with the breeding experiments in progress, evidence furnished by Valleau from Kentucky shows that under certain conditions this blight may become an important factor in limiting production in southern regions. Should further work demonstrate beyond a doubt that the anthracnose lesions referred to on raspberry canes contribute to the spread of the Gloeosporium blight, methods of controlling the blight would involve more effective control of anthracnose by spraying or dusting.

SUMMARY

Gloeosporium blight is reported on red raspberry hybrids in Maryland and Virginia and on commercial varieties of black raspberries in Kentucky, Michigan, Ohio and other states.

The symptoms of the disease are described and differentiated from those of spur blight and blue stem or wilt.

A chromogenic *Gloeosporium* has been isolated from blighted red raspberry canes at Bell, Md., each spring since 1922.

Pure cultures of a non-chromogenic strain of the fungus were obtained by the plating method from a chromogenic culture which was contaminated with bacteria.

According to Shear, the strains of *Gloeosporium* isolated from raspberries cannot be distinguished morphologically from those commonly found on apples and referred to as *Glomerella cingulata* or *Gloeosporium cingulatum*.

Cross inoculations with the chromogenic strain from raspberry and with a chromogenic and a non-chromogenic strain from apple demonstrated the pathogenicity of the three strains on both hosts.

Attempts to infect the raspberry by spraying uninjured canes with spore suspensions indicate that the fungus does not readily penetrate the unbroken epidermis under ordinary growing conditions.

Preliminary studies suggest that anthracnose lesions may open the way for invasion by *G. cingulatum*.

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EXPLANATION OF PLATES

PLATE XXVIII

Gloeosporium blight of raspberry.

A. Portion of cane of Van Fleet raspberry in April, showing effects of natural infection of the growing shoot the previous summer by *Gloeosporium cingulatum*. Black acervuli scattered evenly over the entire surface.

B. Whitish dead streak of tissue on one side of a cane of a red raspberry hybrid at Bell, Maryland, in April. Acervuli were abundant on the dead portion of this cane.

C. Infected main cane. The fungus must have passed down into the main cane after having killed the lateral, a.

D. Leaf stalk of a hybrid red raspberry in the greenhouse ten days after inoculation with a chromogenic strain of *Gloeosporium cingulatum* from apple.

E. Main stem of a young shoot of the plant shown in E. Acervuli developing on the lesion.

F. Black raspberry shoot two weeks after inoculation with the non-chromogenic strain of *Gloeosporium cingulatum* from apple.

A, D, E, and F enlarged.

PLATE XXIX

Gloeosporium blight on cultivated black raspberry received from Dr. W. D. Valleau, Lexington, Kentucky.

A. The blighted tip end (b) and leaves of the main cane. The bluish-purple dead end was covered with acervuli of *G. cingulatum*; at the left, a green branch (a) spotted with anthracnose lesions, each of which also contained acervuli of *G. cingulatum*.

B. Tip end of a blighted young cane split open to show the brown pith with numerous cavities in which a white mycelium of *G. cingulatum* was maturing numerous conidia.

C. Part of the branch (a) shown in A, after it had been cut off and the end placed in water for nearly three days. The upper part had collapsed and turned bluish-black. Large numbers of acervuli were beginning to break through the epidermis. Numerous cavities had been formed in the pith. At the lower end, which was still green, the pith was solid.

A
B
C
D
E
F

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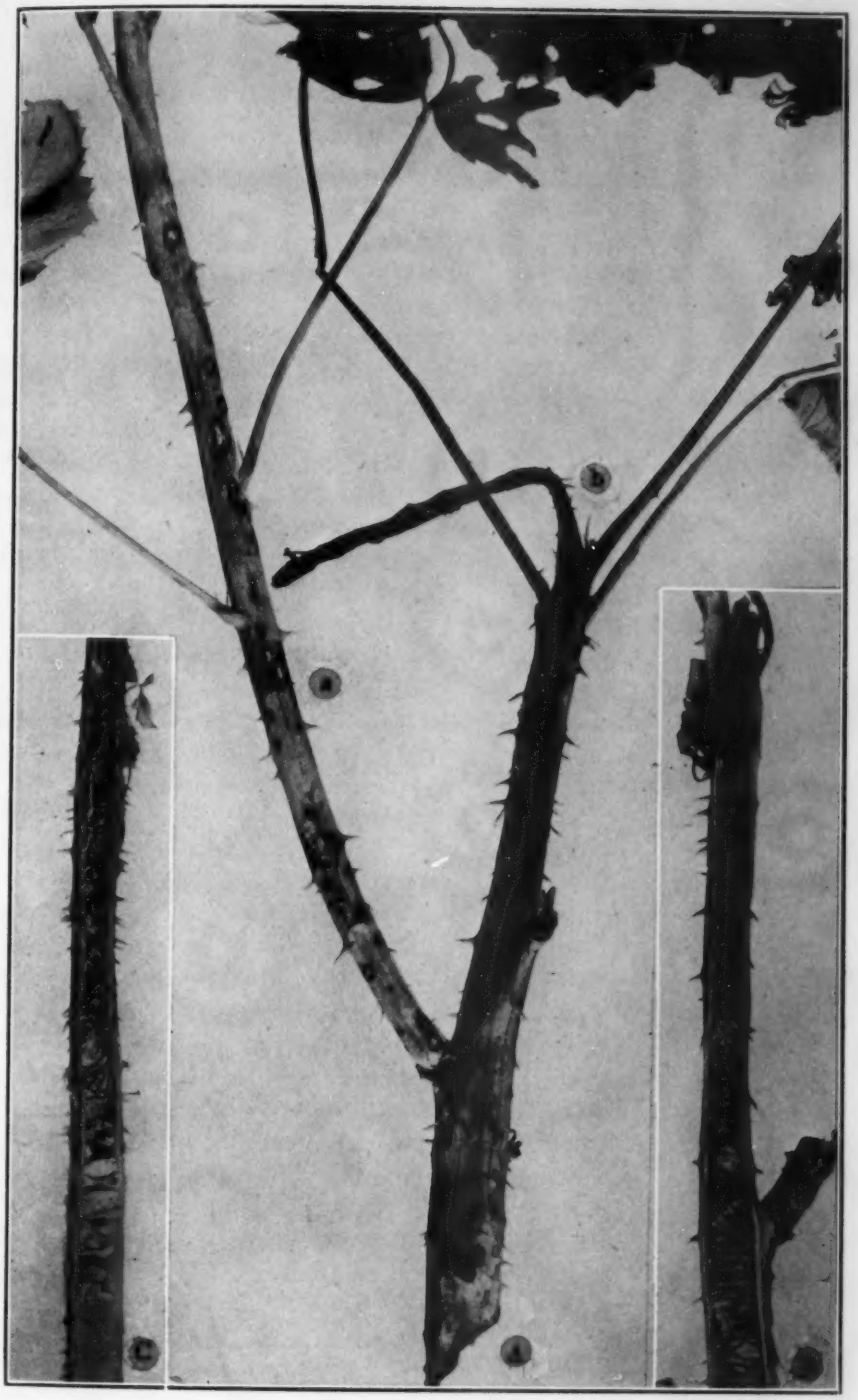

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A POTATO VIRUS ON PEPPERS

F. M. Blodgett

Early in 1925 attempts were made to inoculate pepper plants with the virus of yellow dwarf disease of potatoes. Fortunately, at the same time, inoculations were made on peppers with inoculum from apparently healthy potato tubers and from mosaic and healthy tobacco. On all of the peppers inoculated (February 16) from potato, both from the yellow dwarf tubers and from the healthy tubers, striking symptoms developed (March 2) quite different from those which developed on the plants inoculated with tobacco mosaic virus. The peppers inoculated with inoculum from healthy tobacco remained healthy.

This result was immediately recognized as similar to the then unpublished results of Fernow (1) with inoculations from apparently healthy potatoes to *Nicandra physalodes* (L.) Pers. and to *Nicotiana glutinosa* L., and similar to the results of Johnson (2, 3) with inoculations from potatoes to tobacco. Further work with this disease on pepper has confirmed in many respects the work of the above-mentioned experimenters. In the meantime, in his bulletin, Johnson (2) has listed pepper among the plants susceptible to one or more of the virus diseases which he obtained from potatoes. In a more recent article Schultz (4) has reported finding certain potatoes susceptible to a virus from other apparently healthy potatoes.

The symptoms produced on pepper with the virus from apparently healthy potatoes were so striking that this host was believed to be a favorable one on which to test the theory then just suggested by Johnson in his abstract at the Washington meeting of the American Phytopathological Society, namely, that something in the extract of normal potato foliage causes a disease of tobacco and tomato which is of an infectious nature. Some of Johnson's experimental plants had been seen by the writer, and the results could not be doubted. It was not found easy to duplicate this work, however. Difficulty was experienced in growing tobacco and tomatoes under conditions sufficiently favorable so that the symptoms could be seen, and at best they were not very striking. It seemed particularly pertinent to find out whether the same virus could be obtained from seedling potatoes before these had been infected from commercial potatoes. In other words, if the virus is to be considered as some normal constituent of potato foliage, a necessary condition would seem to be that it should be found in the foliage of seedling potatoes.

In the experiments to be described here, inoculations were made with a flamed needle on which a tuft of absorbent cotton was wound to act as a holder of inoculum. The material to serve as inoculum was ground in a steam sterilized mortar. A fairly regular, although entirely arbitrary, number of punctures was made in each plant: ten in the stem and ten in each of three leaves.

The symptoms produced on peppers by inoculations with the virus from apparently healthy potatoes varied considerably in time of appearance, as in some other mosaics, depending on conditions of growth and temperature. In parallel inoculations they were a few days slower in appearing on peppers inoculated with the virus from potatoes than on those inoculated with tobacco mosaic virus. Under favorable conditions they appeared in ten days.

The first symptoms to be observed are rather indefinite, light spots in

FIG. 1. Pepper plant inoculated from an apparently healthy potato. Necrotic spots have developed in the leaves, and the leaves are more or less distorted.

the young leaves developed after inoculation. These spots quickly die and turn brown. They are variable in shape, and in size up to about a half centimeter in diameter. As the spots are developed in the rapidly expanding younger leaves, these leaves frequently become wrinkled and distorted (Fig. 1). They do not persist on the plants for long, but drop within a few days leaving the plant with only the older leaves developed previous to the virus infection (Fig. 2). Necrotic streaks spreading from points of inoculation are developed frequently also in the stems (Fig. 2). For comparison, a healthy plant is shown in figure 3.

Plants affected as described, however, usually do not die completely. In a short time, after one crop of new leaves has been shed, more buds begin to push out if the plant is kept under conditions favorable for growth. The leaves appear quite normal until about half grown. They then develop lighter colored areas which frequently give them a decidedly mosaic appearance. Soon the light areas become necrotic, the leaves drop, the plant remains dormant for a brief period, and then starts anew the succession of symptoms as described. Such plants naturally remain very much dwarfed. One such plant, however, has remained alive in the greenhouse for over two years.

TABLE 1.—*Results of inoculating tobacco and pepper plants with a virus from different varieties of potato, July 23 to August 4, 1925*

Variety of potatoes used as source of inoculum	Pepper plants		Tobacco plants	
	No. inoculated	No. infected	No. inoculated	No. infected
Green Mountains	4	4	4	4
No. 9	4	4	4	4
Seedlings	4	0	4	0
Cobblers	4	3	4	4
Rural Russets	4	4	4	4
Heavy Weight	4	3	4	4
Bliss Triumph	4	2	4	2
None (check)	5	0	31	0

In this way peppers have become infected in various tests extending over a period of a year and a half. In all, twenty-five lots of pepper plants were inoculated at different times. Of 135 plants inoculated with the virus from various commercial varieties of potatoes, 114 plants became infected and symptoms were as described above. Both punctured and unpunctured checks, 105 in number, have been grown with inoculated plants but have regularly remained free from this disease. In fact, there has been no indication that the disease spreads in the greenhouse.

No extensive search has been made to discover a plant of a commercial variety of potatoes free from this virus, although a few inoculations have

been made from individual potato plants. In March, 1925, fifteen pepper plants were inoculated, each from a different potato plant. Eleven of the fifteen became infected. In a parallel experiment *Nicandra* was inoculated from the same potato plants, and infection was secured from two additional potato plants. In a later experiment, virus from eight Bliss Triumph potato plants was used to inoculate eight sets of five pepper plants each. In these cases the virus from each of the potato plants caused infection in each set of five pepper plants except in one case in which only three of the five became diseased. All the different commercial varieties of potatoes used appear to be carriers of this virus. The results of one experiment in which peppers and tobacco were inoculated with the virus from different varieties of potatoes are given in table 1. It will be noted that all of the varieties except the seedlings proved to be carriers of this virus. The symptoms obtained on tobacco seemed to be of Johnson's "mottle" type and this has been the type regularly obtained from the virus of the potatoes used in these tests where parallel inoculations have been made on tobacco. No differences were noted in the symptoms on peppers inoculated with the virus from these different varieties of potatoes.

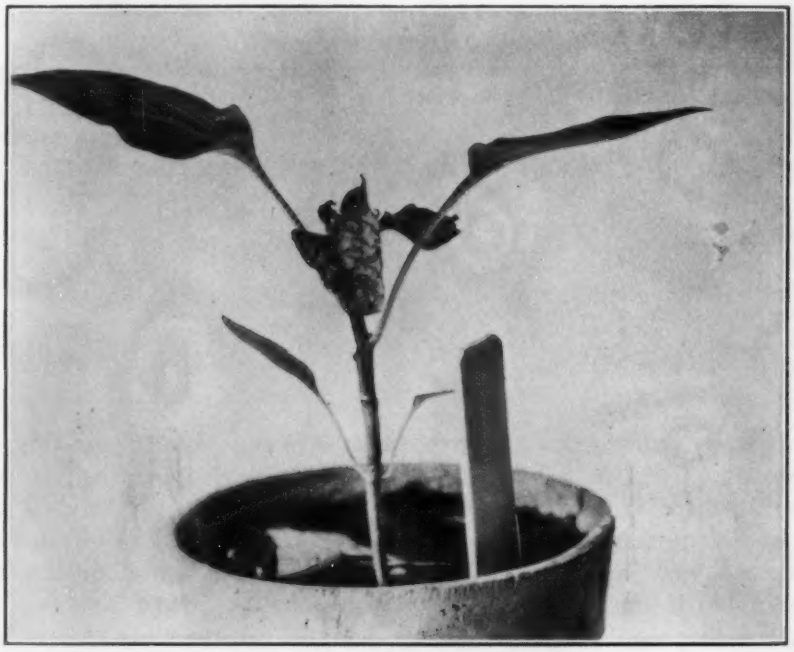

FIG. 2. A little later stage than is shown in figure 1. Most of the affected younger leaves have dropped. The stem shows a necrotic streak.

As noted in one case in table 1, in a number of instances the inoculum was taken from seedling potatoes, *i.e.*, plants grown from true seed. Most of these seedlings were grown from seed produced by the variety Green Mountain. In all, 57 pepper plants were inoculated in 12 different lots with inoculum from seedling potatoes, but in none of these cases did any symptoms develop on the peppers to indicate that a virus had been transmitted.

Seedling potatoes were inoculated with the virus from commercial potatoes at various times, but no symptoms of disease were observed on the seedlings. Three of these were the same ones previously used as a source of inoculum for peppers. The results of inoculating peppers also were negative. Later, at several different times, peppers were inoculated with the virus from these inoculated seedling potatoes. Of 33 pepper plants inoculated, 14 became infected. Equal numbers of pepper plants were inoculated from uninoculated seedling potatoes of the same age and lots in each case but did not become infected. Thus it appears that seedling potatoes, until inoculated, are free from the virus which commonly is carried by the commercial varieties of potato. The percentage of infection on the peppers inoculated from the inoculated seedlings was low, as might be expected in using inoculum from plants with no visible symptoms to serve as guide as to which of the previous inoculations had been successful. The symptoms of this disease on peppers are so striking that no doubt can be entertained in the case of positive infection.

After this potato virus has been transferred to pepper, it may be readily transferred from one pepper to another. When the virus from one of the first peppers inoculated from potatoes was used to inoculate a second lot of 10 peppers, 9 became diseased. A second transfer from one of these peppers to 5 more resulted in all 5 becoming diseased. No difference was noted in the symptoms of the disease on these different lots.

An attempt was made to see whether this potato virus disease is seed-transmitted in peppers. When pepper fruits produced on diseased plants were examined, it was found that they contained very few seed as compared with the fruits on healthy plants. A few seed were obtained, however. They were gathered October 12 and planted November 7, 1925. The seedlings (not counted) raised from these seed all appeared healthy. In addition, 21 of these transplanted to pots continued to look healthy until five of them were inoculated on January 18. Four of these became infected. Although this test was not very extensive, it indicates that the potato virus is not transmitted through pepper seed at least to any considerable extent, and that plants raised from seed produced on diseased plants are still susceptible to the potato virus disease.

No symptoms were detected when Bliss Triumph potatoes were inoculated with the potato virus from diseased peppers. Fifty potato plants were inoculated in April, 1925, with the potato virus from diseased peppers. These were kept until they died in July. Parallel inoculations were made with material from healthy peppers, the potato virus (Johnson's "mottle") from tobacco, and mild mosaic of potatoes. A large number of uninoculated plants was kept for comparison. No symptoms of disease were observed on any of these plants. When some of the progeny were grown, the only disease which developed was mild mosaic. Results were as follows: 3 plants with mild mosaic of 39 inoculated with potato virus from pepper, 0 out of

FIG. 3. A healthy pepper plant of the same age as those shown in figures 1 and 2.

27 inoculated with potato virus from tobacco, 10 out of 18 inoculated with mild mosaic, and 2 of 34 uninoculated. While the results of this series of inoculations are unsatisfactory, showing as they do only low percentages of infection when inoculations were made with the virus of mild mosaic and probably a natural spread of mild mosaic to some extent in the whole series, certainly no striking necrotic symptoms were secured. So far as this evidence goes, it indicates that the disease in question in this work was not Johnson's "spot-necrosis," but it does correspond with his "mottle."

So far, only a few plants having this disease have been seen in commercial fields, but only a few fields of peppers have been examined since these experiments were begun. How important the disease is commercially is not known. It might naturally be expected to occur in peppers when planted near potatoes, especially if an insect carrier is present.

SUMMARY

A serious disease of peppers is caused by inoculating them with the virus from apparently healthy commercial varieties of potatoes. This is apparently caused by the same virus mosaic B from potatoes that Fernow has used in inoculating *Nicandra physalodes* and *Nicotiana glutinosa* and some six other hosts, and which Johnson has described as the cause of "mottle" on tobacco and which he has shown may be transmitted to some thirty species of plants.

The symptoms of the "mottle" potato mosaic on pepper are marked. Faint mottling develops on leaves about half grown. The light colored areas quickly become necrotic, and affected leaves drop shortly. Necrotic areas spreading from inoculation points frequently develop on the stems. The plants are dwarfed. The only potatoes found from which the potato "mottle" virus could not be obtained were seedling potatoes from true seed. The virus could be transmitted to such seedlings by inoculation.

The potato virus is transmitted readily from one pepper to another by inoculation.

The virus is neither transmitted through pepper seed nor potato seed, although it is transmitted through potato tubers.

The extent of the occurrence of this disease in commercial fields is not known.

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PHYSIOLOGIC SPECIALIZATION IN PUCCINIA CORONATA AVENAE

H. E. Parson[1]

INTRODUCTION

In 1919 Hoerner (3) demonstrated that there are physiologic forms of *Puccinia coronata avenae* (Corda) Erikss. and Henn. which differ in their pathogenicity on certain varieties of oats. There has been but little, if any, subsequent investigation of the problem. In fact, Hoerner's results never have been amplified, or even confirmed. And, as far as the writer knows, no attempt has been made to take into consideration the established fact of physiologic specialization in developing varieties of oats resistant to crown rust.

It is quite likely that crown rust causes greater aggregate losses to oats in the United States than does stem rust. In some of the southern oats-growing sections crown rust is very destructive while stem rust is relatively unimportant. In the upper Mississippi Valley crown rust often causes heavy losses in the vicinity of buckthorn bushes; and sometimes, as in 1927, general epidemics develop and reduce yields greatly.

Something should be done to reduce losses from crown rust. In many of the northern oats-growing states the eradication of buckthorn bushes would reduce losses considerably, but this would not be true of the Southern States. Even if the eradication of the buckthorn would reduces losses greatly in the Northern States, it is doubtful whether it would be feasible to organize and successfully complete an extensive eradication campaign. And the writer is convinced from personal observation that the buckthorns will remain and multiply forever unless such a campaign is begun soon. Crown rust will become increasingly destructive in the Northern States as the number of buckthorn bushes increases, and the rust will continue to take its annual toll in the Southern States—unless resistant varieties of oats are produced.

The development of varieties of oats resistant both to crown rust and stem rust may not be easy. It would be most desirable to breed varieties

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The writer wishes to acknowledge his indebtedness to Dr. E. C. Stakman for suggestions and criticism during the progress of the work and for help in preparation of the manuscript.

resistant to the two rusts, the smuts, and spikelet sterility. Anthony (Minn. 686) derived from White Tartar × Victory has been resistant to stem rust in many widely separated localities in the United States, but it is very susceptible to crown rust and to the smuts. Markton (C. I. 2053), on the other hand, is very resistant to the two smuts, but susceptible to the rusts. The whole problem is complex because there are physiologic forms of *Ustilago avenae* and *U. levis* (5), of *Puccinia graminis avenae* (1, 9) and *P. coronata avenae*. However, as the smuts can be controlled by seed treatment, it would be a valuable contribution to develop varieties resistant to the two rusts. If this contribution is to be made, the physiologic specialization of the rusts must be investigated and the facts obtained must be used in breeding work. For this reason the present study was made.

MATERIALS AND METHODS

One way of determining whether there are physiologic forms of a pathogenic fungus is to inoculate varieties ordinarily resistant with collections from different places. If the varieties are susceptible to any of the collections, it is evidence of the existence of forms. On the other hand, if any usually susceptible varieties are resistant, there also would be evidence of the existence of forms. In the present work the writer selected a number of varieties of oats which had been reported as resistant to crown rust (2, 4, 6). These were inoculated with collections of rust obtained from fifteen different places in the United States and Canada.[2]

Most of these collections were from cultivated oats, but a few were obtained from Rhamnus spp. The rusted material was sent to University Farm, St. Paul, and inoculations were made as soon after arrival as possible.

The inoculation technique was similar to that described by Stakman and Piemeisel (7). Victory oats was used for stock cultures, although Richland was used in a few cases.

RESULTS

It was quite evident that there were distinct differences in the pathogenicity of the different collections of rust obtained. The types of infection described by Stakman and Levine (8) for *P. graminis tritici* all appeared with the exception of type 2. Type 3 sometimes approached type 2, but no clear-cut type 2 was ever observed. For convenience, the descriptions of these types are given below.

[2] The writer wishes to acknowledge his indebtedness to the pathologists and agronomists who were kind enough to send rust and seed material, and to T. R. Stanton, Office of Cereal Crops and Diseases, United States Department of Agriculture, for seed of some of the varieties of oats.

"0—*Immune*

"No uredinia developed; hypersensitive flecks usually present, but sometimes there is apparent absolutely no trace of mycelial invasion in the host tissues

"1—*Very Resistant*

"Uredinia minute and isolated; surrounded by sharp, continuous, hypersensitive, necrotic areas

"2—*Moderately Resistant*

"Uredinia isolated and small to medium in size; hypersensitive areas present in the form of necrotic halos or circles; pustules often in green, but slightly chlorotic, islands

"3—*Moderately Susceptible*

"Uredinia medium in size; coalescence infrequent; development of rust somewhat subnormal; true hypersensitiveness absent; chlorotic areas, however, may be present

"4—*Very Susceptible*

"Uredinia large, numerous and confluent; true hypersensitiveness entirely absent, but chlorosis may be present when cultural conditions are unfavorable."

Plants were considered resistant if they developed a type 1 infection. If they developed a type 3 or type 4, they were considered susceptible. In the determination of forms, resistance and susceptibility only were taken into consideration.

The results of the inoculations are summarized in table 1. It shows clearly that the different collections naturally fall into several groups with respect to their pathogenicity. All of the varieties inoculated are susceptible to the collections in group 1. The rusts in this group, therefore, are considered to be form 1. In the second group all varieties are susceptible, except Green Mountain, which is resistant. The difference between collections in this group and those in the first group is simply in their effect on Green Mountain. The collections in this second group are considered to be form 2. *Avena sterilis nigra, A. sterilis* selection, Burt, Red Rustproof, and Ruakura are all resistant to form 3, while Green Mountain is susceptible to this form. Ruakura is the only variety resistant to form 4. The four forms are very distinct. In fact, the differences in their pathogenicity are so sharp that one is forced to conclude that they are distinct physiologic forms. The differences are quite as pronounced as are the differences between physiologic forms of *P. graminis tritici.* There is a possibility that there is a fifth form. The collection obtained from Ste. Anne de la Pocatiere, Quebec, is quite similar to form 3, except for the fact that *A. sterilis nigra* and *A. sterilis* selection are moderately susceptible to the Ste. Anne collection instead of being resistant. There is a wide difference in the reaction of these varieties to the two rusts, and it seems justifiable, therefore,

TABLE 1.—*Reaction of 27 species, varieties, and selections of Avena to Puccinia coronata avenae collected in various places in the United States and Canada in 1926 and 1927*

Varieties of oats inoculated	Place of collection and rust reaction														
	Group 1								Group 2			Group 3		Group 4	Group 5
	A and M. College, Miss.	Chatham, Mich.	Columbia, Mo.	Denton, Tex. (Collection No. 20)	San Antonio, Tex.	Stillwater, Okla.	Tifton, Ga.	Yankton, S. Dak.	Durant, Okla.	Lincoln, Nebr.	Mill Creek, Okla.	Omaha, Nebr.	St. Paul, Minn.	Denton, Tex. (Collection No. 11)	Ste. Anne de la Pocatiere, Que., Can.
Green Mountain	S	S	S	S	S	S	S	S	R	R	R	S	S	S	S
A. sterilis nigra[a]	S	S	S	S	S	S	S	S	S	S	S	R	R	S	MS[b]
A. sterilis selection[a]	S	S	S	S	S	S	S	S	S	S	S	R	R	S	MS[b]
Red Rustproof	S	S	S	–	S	–	S	–	S	S	S	R	R	S	R
Red Rustproof, C. I. 1039	S	S	S	–	S	–	S	–	S	S	S	R	R	S	R
Red Rustproof, C. I. 1815	S	S	S	S	S	S	S	S	S	S	S	R	R	S	R
Ruakura, C. I. 2025	S	S	S	S	S	S	S	S	S	S	S	R	R	R	R
Burt	S	S	S	S	S	S	S	S	S	S	S	R	R	S	R
Burt, C. I. 2042	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S
Burt 916, C. I. 2054	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S
Avena barbata	–	–	–	–	S	–	–	–	–	S	S	–	S	–	–
A. brevis, colored	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S
A. brevis, white	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S
A. nuda	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S
A. sterilis ludoviciana	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S
A. strigosa	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S
C. I. 606	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S
Culberson	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S
Culred, C. I. 518–189	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S
Fulghum, C. I. 708	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S
Green Russian	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S
Kanota	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S
Kanota, C. I. 839	–	–	–	–	S	–	–	–	S	–	–	–	S	–	–
King	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S
Rustless, C. I. 724	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S
White Russian, C. I. 1871	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S
Winter Turf[c]	S	S	S	–	S	–	S	–	S	S	S	S	S	S	S

[a] There was some evidence on the basis of the reaction to the rust that these were not pure lines.

[b] The uredinia were fairly large but were surrounded by pronounced necrotic areas.

[c] The varieties here listed without numbers are those of Etheridge, obtained through the Agronomy Department of the University of Minnesota.

to conclude, at least tentatively, that the Ste. Anne collection represents a fifth form.

The differences in the infection ability of these five forms are shown more clearly in table 2. In this table are listed only the four varieties which served as differential hosts.

TABLE 2.—*Reaction of four differential varieties of oats to five physiologic forms of Puccinia coronata avenae*

Variety	Physiologic form and place of collection[a]				
	1	2	3	4	5
	San Antonio, Tex.	Durant, Okla.	St. Paul, Minn.	Denton, Tex.	Ste. Anne de la Pocatiere, Que., Canada
Avena sterilis nigra	S	S	R	S	MS
Red Rustproof, C. I 1815	S	S	R	S	R
Ruakura, C. I. 2025	S	S	R	R	R
Green Mountain	S	R	S	S	S

[a] The writer is indebted to the following for the collections of rust: to Wallace Butler for Nos. 1 and 2; to P. B. Dunkle for No. 4; to H. N. Racicot for No. 5. No. 3 was obtained from stock cultures growing in the Plant Pathology greenhouses at University Farm, St. Paul.

The writer has made an analytical key for the identification of the five physiologic forms of *Puccinia coronata avenae* within the genus *Avena*:

Ruakura—resistant (Hoerner's Form 3)
  Avena sterilis nigra—resistant ..... Form 3
  A. sterilis nigra—susceptible
    Red Rustproof—resistant ..... Form 5
    Red Rustproof—susceptible ..... Form 4
Ruakura—susceptible (Hoerner's Form 1)
  Green Mountain—resistant ..... Form 2
  Green Mountain—susceptible ..... Form 1

The question naturally arises as to whether some of the forms which the writer isolated were identical with those which Hoerner described. Hoerner used Ruakura and Green Russian as differential hosts. He distinguished four forms as follows:

Form 1. Infects both Iowa 73 [Ruakura] and Iowa 96 [Green Russian] normally.

Form 2. Infects both varieties weakly.

Form 3. Infects Iowa 73 weakly but infects Iowa 96 normally.

Form 4. Infects Iowa 73 normally but infects Iowa 96 weakly.

As Green Russian was susceptible to all of the collections of rust which the writer tried, it is evident that Hoerner's forms 2 and 4 were not found in the present study, unless the Green Russian material used by the writer was different from that used by Hoerner. There is a possibility that Hoerner's form 1 could be either identical with the writer's form 1 or 2, or might be separated into forms 1 and 2 described in this paper. Furthermore, if the collection from Ste. Anne is a fifth form, as it appears to be, it would be possible for Hoerner's form 3 to be either form 3, 4, or 5 described in this paper, or consist of a combination of any two of these forms, or all three of them (see key).

It is entirely possible, of course, that the forms described in this paper could be sub-divided still further if more varieties of oats were inoculated. It may be possible also that other differential hosts would prove better than those which the writer used. It is likely that there are a great many physiologic forms, and the chance of detecting them simply depends upon making many inoculations on many different varieties of oats, and possibly on wild grasses.

Some of the physiologic forms differ from each other in the readiness with which they produce telia. Some of the forms never produced telia during the entire course of the experiment, while others consistently produced them about three weeks after inoculation. Parker (4) was of the opinion that early production of the telia was indicative of resistance of the host plants. However, in the writer's experiments, telia were formed by certain forms as early on susceptible varieties as on resistant ones. All of the writer's experience indicates that the early production of telia is due to an inherent tendency of a particular physiologic form of rust rather than to resistance of the host plants. Form 3 showed a marked tendency to produce telia. This form infects fewer varieties than any of the other forms. It is possible that the early formation of telia is characteristic of narrowly specailized forms. However, further investigations are needed before this conclusion can be drawn finally.

CONCLUSIONS

There can be no question that there are physiologic forms of *P. coronata avenae*. Hoerner was able to recognize four on the basis of their effect on only two varieties of oats. The writer has been able to recognize five by their effect on four differential hosts of Avena spp. The types of infection produced by these different forms are distinct and constant. It is rather remarkable that all of the varieties inoculated are susceptible to form 1, and

all but one (Green Mountain) are susceptible to form 2. All of the differential hosts are resistant to form 3 except Green Mountain, which is susceptible. These differences have been consistent after repeated inoculations. There can be no question but that these physiologic forms differ from each other genotypically. Further proof of differences is the fact that some forms have a tendency to produce telia earlier than others. This tendency is quite independent of the resistance or susceptibility of the host but seems to be a genetic characteristic of the physiologic form of rust. It seems likely that there is a correlation between the narrow pathogenic specialization of a strain and early formation of telia, but this cannot yet be stated with certainty.

Crown rust often causes heavy losses both in the Southern States and in the Northern States. In the South the urediniospores apparently survive the winter and the rust is independent of the alternate host, the buckthorn. In the Northern States, however, there seems to be abundant evidence that destructive epidemics of crown rust usually occur only near buckthorn bushes. However, in 1927 a widespread and destructive epidemic developed. It is doubtful whether this could have been traceable wholly to buckthorns. The only solution of the crown rust problem in the South would seem to be the development of resistant varieties. While the eradication of the buckthorns from the Northern States undoubtedly would reduce losses in most years, it is doubtful whether the eradication of the bushes could be successfully accomplished. Therefore the development of resistant varieties for the Northern States also is desirable. In order that such breeding work may be placed on a sound basis, it would seem highly desirable to make an extensive study of the number, distribution, and pathogenic capabilities of the physiologic forms of *P. coronata avenae*.

SUMMARY

1. The writer obtained collections of *P. coronata avenae* from fifteen different places in the United States and Canada. Twenty-seven varieties, selections, and species of cultivated and wild oats were inoculated with these collections.

2. Four varieties of oats proved to be differential hosts. On the basis of the reaction of these varieties to the different collections of rust, it was possible to distinguish four, and possibly five, physiologic forms. It is very likely that many other forms can be recognized if more extensive work is done.

3. Physiologic forms of *P. coronata avenae* differ in their effect on host plants. In some cases the effect on a single host determines the identity of the form.

4. The physiologic forms apparently differ also in the readiness with which they produce telia. The production of telia seems to be independent of the resistance or susceptibility of the variety on which the rust is growing. It seems rather to be due to an inherent tendency within the forms.

5. In developing varieties of oats resistant to crown rust, it will be necessary to take into consideration the physiologic specialization of the pathogene.

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PHYTOPATHOLOGICAL NOTES

Two Fungi on Sclerotinia Apothecia.—In the course of experiments with apothecia of the peach brown-rot fungus, *Sclerotinia americana* (*S. fructicola*), two other fungi were at times prominent as disturbing elements. They were seen first, in the spring of 1921, on apothecia developing from brown-rot mummies which were collected in a local peach orchard and kept in the greenhouse at 10–22° C. When first noted, about a third of the apothecia present were coated with a dense white mycelial growth. This spread rapidly, attacking small developing apothecia as well as the larger mature ones. The apothecia in one box were not infected and were still actively sporulating after the diseased apothecia had dried up. Upon microscopic examination two fungi were found, the most common of which was a *Fusarium* and the other a *Trichoderma* (*T. lignorum* or *koningi*).[1]

In the spring of 1922 inoculations were made with cultures saved from the previous year and Sclerotinia apothecia grown from mummies gathered in the orchard after the developing apothecia were already visible. The apothecia were allowed to develop thereafter in the laboratory, using the method employed for physiological experiments with apothecia in which peach mummies were suspended by chromel wire in tumblers half filled with distilled water, the bottoms of the mummies dipping into the water.[2] To keep the atmosphere saturated, each tumbler was covered with a dish. In the present experiment the room temperature varied daily from about 15° to 30° C. Under these conditions Sclerotinia apothecia grow readily and apparently quite naturally.

Two peach mummies, each bearing apothecia ranging from mature ones to those that had not yet formed disks, were used for each of the organisms. A bit of material from the culture was deposited lightly on one apothecium per mummy in each case. Both *Fusarium* inoculations were plainly successful in two days, a heavy, white growth of *Fusarium* hyphae showing on the inoculated apothecia. In two more days the fungus had spread to other apothecia (Fig. 1), and three days later every apothecium on the inoculated mummies was obscured in a mass of mycelium. The *Fusarium* was reisolated readily from these diseased apothecia. No infection occurred on apothecia inoculated with the *Trichoderma* or on the checks.

[1] Cultures of these organisms were lost by mite infestation before more definite identification was attempted.

[2] Norton, J. B. S., W. N. Ezekiel, and R. A. Jehle. Fruit-rotting Sclerotinias I. Apothecia of the brown-rot fungus. Md. Agr. Exp. Sta. Bul. 256: 3–32. 1923. (See figs. 17 and 18.)

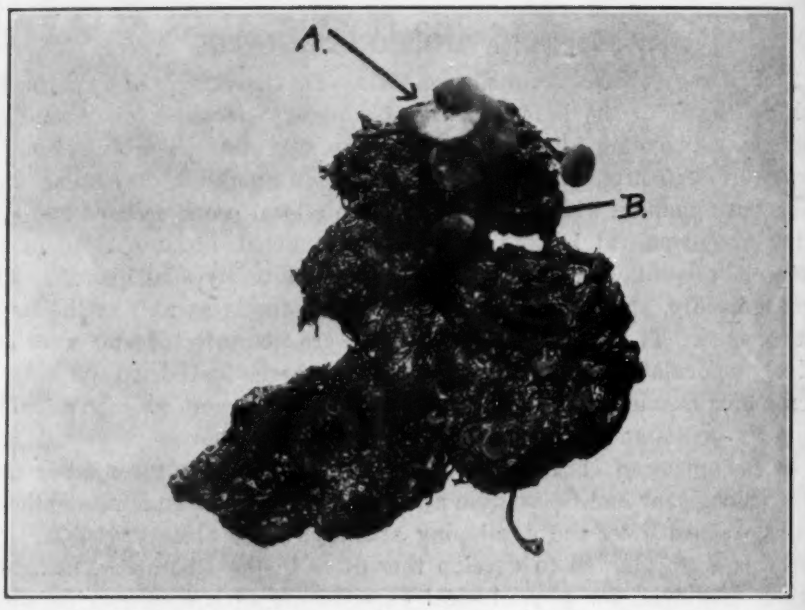

FIG. 1. *Fusarium sp.* on apothecia of *Sclerotinia americana*. Photographed four days after apothecium (A) was inoculated. Note hyphal spread as far as (B). ×1 1/3.

Both organisms were frequently found in the spring of 1922 on apothecia growing under experimental conditions such as those described above. The *Trichoderma* was observed chiefly on apothecia injured by the various toxic substrata to which they had been exposed; while the *Fusarium* was found on vigorously growing apothecia also, in agreement with the inoculation results.

Macroscopically the two fungi may appear rather similar on apothecia until spores are produced, when the dark green, mound-like masses of the *Trichoderma* are readily distinguished from the pinkish *Fusarium*.

The writer has never observed this fungus injury to apothecia in the field. Even though these organisms seem not uncommon on peach brown-rot mummies, it is unlikely that even the *Fusarium* is of any considerable importance in the destruction of apothecia under natural conditions. Fortunately, the discovery or dissemination of such natural enemies of this *Sclerotinia* has little practical significance, as the dangerous source of brown-rot infection from apothecia can be controlled otherwise, for instance by the old practice of burying mummies by plowing or other cultivation, which not only prevents apothecial development but also hastens disintegration of the mummies themselves.[3]—WALTER N. EZEKIEL, Maryland Agricultural Experiment Station, College Park, Md.

[3] Ezekiel, Walter N. Fruit-rotting Sclerotinias III. Longevity of buried brown-rot mummies. Md. Agr. Exp. Sta. Bul. 284: 9–22. 1926.